

THE SYMPTOMS, DIAGNOSIS, PATHOLOGY AND
TREATMENT OF SHIP BERI-BERI.

With a review of the various theories in
regard to the Etiology.

Beri-Beri is the name applied to a specific form of multiple peripheral neuritis, the symptoms or secondary manifestations of a primary microbial infection, or the absorption of a toxin acting on the Nervous System producing well marked signs. Manson defines Beri-Beri as "a form of Peripheral neuritis which occurs endemically and epidemically and is especially characterized by proneness to oedema and to implication of the neuromuscular System of the central organs of circulation by complete absence of trophic skin lesions, of paresis of the muscles of the head and neck..... There are troubles of locomotion parasthesia of various

description, especially in the lower extremities. Marked hyperaesthesia of the muscles involved with subsequent atrophy etc."

There are few diseases the etiology or Primary cause of which is so much based on hypothesis and theories as that of Beri-Beri, and on reading over the many and varied suggestions given by different observers, one comes to the conclusion that the disease is caused directly by the introduction into the system, by food or drink, of a specific organism as yet undetermined, or indirectly by the absorption or inhalation of a toxin derived from such an agent . This applies more however to cases of true or tropical Beri-Beri, as Ship Beri-Beri seems from the History and Symptoms presented to differ in some respects from ordinary Beri-Beri and indeed, as I shall endeavour to show, is

believed by many who have closely studied cases of Ship Beri-Beri, to be two distinct diseases, though allied features are presented in both.

The geographical distribution of the disease is pretty general. It is to be found in India, China, Japan, Malay States, Central America, South America, West and Central Africa, Norway and Sweden. Though considered among tropical diseases it is really not so, as the disease is fairly common in sub-tropical and temperate climates.

Before considering the various theories put forward with regard to the etiology of Beri-Beri I propose first, to give the symptoms and Clinical features presented by typical cases of Ship Beri-Beri and contrast these with those presented by cases of true or tropical Beri-Beri. Pekelharing and Winkler describe the initial

stages of Beri-Beri as "Slight oedema along the crest of the tibia, a puffy, pasty face, difficulty in certain movements observable only at first when the patient walks quickly or endeavours to go upstairs; some parasthesia or anaesthesia of the lower extremities; palpitation of the heart, a slightly quickened pulse, or rather a pulse which remains within normal limits when the patient is at rest, but which upon the least exertion goes up to ninety or a hundred beats per minute, a marked contrast between the violent beats of the heart and the small and feeble pulse, a prolonged first and reduplicated second sound of the heart."

To illustrate and follow out the Signs and Symptoms given above, on individual cases, I submit the following notes on a few of the cases of Ship Beri-Beri treated at the Seamen's

Hospital, Greenwich, to show that in the main, the symptoms are similar in all, though slight variations from the typical occasionally occur.

Case 1.

A.H. an able seaman admitted into the Seamen's Hospital, complaining of pain in legs, chest and stomach.

History: Patient was an A.B. on a sailing ship on a voyage from Calcutta to London. The voyage took four months and all went well until about one month before arrival, when two of the older hands got ill, with swelling and loss of power in both lower limbs, feeling of extreme weakness and lassitude. These two patients died in less than three weeks. A few days after they got ill two others began to complain. Each had swelling starting in the legs, coming on suddenly, and they all got very short-winded.

The swelling gradually worked upwards to the thigh and on to the abdomen. ~~t~~he arms and side of face, but not the eyelids. They soon had to give up work, but they never completely lost the power of the legs, but once or twice felt as if the legs would give way under them. (Two of the men passed blood by the bowel.) The swelling in the legs begun to subside at the end of a week, but recurred again in a few days' time, but subsided in the course of 24 hours, leaving the patient very weak and helpless.

The food consisted mainly of salt beef and pork. No fresh meat and hardly any vegetables.

Condition on Examination.

Patient fairly healthy-looking man, no oedema or cyanosis. Finger nails not clubbed. Well developed, but says he has lost much weight and the calf muscles are much more swollen than they were before he took ill.


Circulatory System

Apex beat 5th interspace just outside the left mid clav. line, feeble impulse.

Rt. Border $\frac{3}{4}$ " to rt. of rt. Sternum margin

Lt. " $\frac{1}{4}$ " beyond left. mid. clav. line.

Auscultation

Systolic murmur at apex 

First sound at ^{Base} ~~apex~~ reduplicated

Second sound sharp and clicking.

Knee Jerks brisk, no ankle clonus. Muscular hyperaesthesia in legs and calves, but not in arms. Spleen and liver normal, urine normal.

Detained in Hospital two months. Discharged convalescent.

Case 2.

L.C. aged 62. (Norwegian.)

Complaint: Loss of power in legs.

History: Never had Syphilis. Had Gonorrhoea once. Ague twenty years ago.

Moderate drinker. Has never had a fit of any sort. Present trouble first appeared in October 1907 at Hull, when patient had returned a week previously from a voyage to Maine (U.S.A.) He had some pain and tenderness in the ankles and calves of the legs. The trouble at first was slight and patient took a ship to Boston but got much worse on the voyage. His legs became swollen and his arms and hands got weak and painful. On arrival at Boston patient went into a Hospital and was told he was suffering from Beri-Beri. He was the only member of the crew ill on the voyage.

State on admission: Slight oedema of lower extremities as far as the knees. Complains of pins and needles in his toes. Considerable loss of power in upper and lower extremities and much muscular wasting with a tendency to foot-drop.

Knee jerks absent. Urine normal.

Circulatory System.

Pulse tension rather raised. Some atheroma of peripheral arteries. No Bruits. Ronchi and Ryles in chest. No cardiac enlargement. Temperature subnormal all the time. Detained in Hospital 6 months. Discharged much improved.

Case. 3.

L.H. aged 32. (English).

Admitted complaining of swelling and pain in legs and arms. Duration of illness - three weeks. Never had a previous attack.

Previous History: Malaria and Scarlet Fever.

Has just returned from San Francisco without calling anywhere. Previous to that he had been to Central America and to the Philippine Islands.

C.O.A: Temperature 99. Pulse 88. Respiration 28. Slight Diarrhoea.

There is great oedema of both thighs and legs, and to a less extent of both forearms, also in the lumbar region. Ten days ago he also had oedema of face. No defect of vision. No anaesthesia. The limbs feel sore. Knee Jerks absent. No ankle clonus. Plantor reflex normal.

Heart: Apex beat nipple line. Pulmonary Systolic murmur. Oedema of chest and abdomen. Temperature normal. Urine normal.

Case. 4.

Ling. aged 29. Chinaman.

Complaint: swelling in the legs, pain in the abdomen and constipation.

Condition of Examination: Patient well formed and nourished. Temperature normal. There is general anasarca especially marked on legs and abdominal wall, and over base of Sacrum. Patient is dyspnoeic and lips are

livid. Pulse regular. Good tension.

Heart: Apex Beat internal to Nipple line.

No thrill. Sounds loud and distinct. No bruit

heard. No distension of Right side of heart.

Lungs: A few crepitations at bases.

Abdomen: Somewhat distended. No sign of

ascites. Skin slightly oedematous all over.

Pain and tenderness in epigastrium where skin is

red and inflamed. Hepatic and Splenic dullness

slightly increased. Tongue clean and dry.

Limbs: India-rubber like oedema of legs,

feet and thighs, with some pitting on pressure.

No marked loss of power. Sensation impossible

to ascertain.

Reflexes: Patella and plantar absent.

Gait: Normal.

Urine: Specific Gravity 1020. Albumen -

trace. Acid.

Patient died two days after admission. (I shall refer to the autopsy later.)

Case 5.

G.I. Norwegian. Admitted complaining of pain and weakness of legs. Patient arrived at Falmouth on a sailing ship, on which were seven other cases of Beri-Beri, two of which ended fatally. Patient has been ill for 24 days, with pain in the side and loss of appetite, and great swelling of the legs. He was confined to bed for some time and on trying to get up he was unable to walk, owing to the weakness of his legs. Ten days before admission his legs got very painful. On admission legs were tender to pressure all along the thighs and calves. Knee Jerks gone. No ankle clonus.

No cardiac enlargement. Heart sounds feeble. Temperature 97.

Detained in Hospital 6 weeks. Discharged cured.

Case 6.

A.S. Chinaman.

Complaint: Pain in legs. Duration 1 month.

Condition on Examination: Oedema of legs,

back and Sternum. Genitals normal.

Pain on pressure on nearly all the muscles of the body, but especially pectorals. Passing small quantities of urine. No albumen. No vomiting. Pulse 120.

Heart: Increased precordial dullness to right of sternum. Liver and spleen normal. Systolic murmur, all over precordium. Patient unable to walk. Gait uncertain. Lungs normal. - *Knee jerks absent* -

No effusion into chest or pericordium.

Case 7.

T. G. aged 26. Norwegian.

Complaint: Swelling of the legs.

History: Patient was on a voyage from Cuba on a sailing ship. This took about two months. About 11 days ago his legs started to swell and patient began to feel weak, but he had no pain. There were two others on the same vessel similarly affected, one of whom died.

State on admission: Patient looks fairly well. Nails slightly clubbed. Oedema of feet and lower legs. No Cyanosis.

Circulatory System:

Pulsation visible on the 4th interspace just inside left nipple line. No bulging of precordia: on palpation this is felt to be slightly wavy and to extend to the 5th space. Suspicion of a thrill. No other pulsation felt.

Percussion: Rt. Border 4th space $\frac{1}{2}$ " to rt.

of sternum.

Lt. Border at left mid clav.

line.

Auscultation: No murmurs heard. Rythm
tic-tac with marked shortening of second pause.
Both sounds very short and sharp.

Pulse regular in time and force. Rise and
fall both rapid. Tension low, walls slightly
thickened.

Respiratory System: Nil

Alimentary System: Nil.

Nervous System: Knee jerks absent. No
Babinski.

Oedema of both legs. Detained in Hospital $2\frac{1}{2}$
months. Discharged cured.

Case 8.

G. H. aged 32. Swede.

Complaint: Weakness of the legs.

History: On return voyage from Singapore was
attacked with weakness and pain in the legs. Feet
and legs were much swollen. Eleven other members

of the crew were similarly attacked and four died.

Condition on admission:

No pain, tenderness or swelling of lower extremities. There is however considerable loss of power of both legs. Both knee jerks are absent.

Circulatory System: Soft blowing Systolic mitral. Cardiac area - normal. Temperature and pulse normal.

Detained in Hospital three weeks. Discharged cured.

Case 9.

W.T. Chinaman, aged 26.

Complaint: Pain and swelling of legs and soreness of chest.

History: Four weeks ago patient first noticed pain in the legs (then at Singapore). Three weeks ago he was unable to work. One week

Temperature normal. Pulse 80 and 100 per minute.

Detained in Hospital 16 days. Discharged cured.

Case 10.

R. T. aged 26. Englishman.

Complaint: Weakness in both legs.

Patient never had venereal disease. Present trouble came on sixteen days ago, when three months out on a return voyage from Peru. The weakness of the legs was severe and patient was unable to walk. Right hand and arm also affected. He had considerable swelling in legs and feet. Two other members of the crew were similarly affected. Patient also suffers from shooting pains round the heart.

State on admission: There is no oedema of legs or feet. Patient complains of tenderness in both calves. Knee jerks absent. Plantar reflex - flexor. Sensation to touch over lower extremity is delayed. There is very marked loss

of power in both legs and in Right arm.

Circulatory System: Apex beat 4th interspace $\frac{1}{2}$ " inside mid. clav. line. A.C.D. normal. No murmurs. Patient complains of numbed feeling in the pit of the stomach and says for the first four days of his illness he had a lot of vomiting. He also feels as if a rope were tied tightly round his waist. Pupils normal. Temperature Pulse and Respiration - normal.

Case 11.

J. H. 38. Englishman.

Complaint: General weakness.

History: Four weeks ago patient's legs started to swell and he felt very weak. Then swelling went down and started again. Did not go into arms or abdomen, but was in chest and back.

State on admission: Patient very weak. No

dropsy or cyanosis. Mouth very filthy.

Heart: Right Border 1" to right of sternum.

Left Border about $\frac{3}{4}$ " beyond ~~E~~ mid

clav. line.

A.B. 5th space. vigorous.

Auscultation: Reduplicating 1st at apex.

Lungs and Abdomen: Healthy.

Knee jerks absent. Muscular tenderness on
calves and thigh. No tenderness over tibia.

Urine normal. No albumen.

Temperature and pulse normal.

Detained in Hospital 2 weeks. Discharged
convalescent.

Case 12.

S. J. Norwegian.

Complaint: Weakness of legs.

History: Patient was on a voyage from
Christiania to South Africa and back and he felt

ill in Delagoa Bay and was taken to Hospital from weakness of legs.

Condition on admission: Patient pale and sallow. No dropsy or cyanosis.

Nervous System: Knee jerks absent. No ankle clonus. No Babinski.

Great muscular hyperaesthesia in both legs. No weakness of upper limbs.

Circulatory System: Heart slightly dilated Systolic apical murmur. Treated by Faradic current.

Temperature, Pulse and Respiration normal.

Detained in Hospital 10 weeks. Discharged cured.

Case 13.

F. W. Englishman.

Complaint: Pain and weakness of the legs.

History: Five months ago patient was in the Southern State of America and had malaria there. He left there for Buenos Ayres and on board ship was attacked with pain and weakness in both legs

and palpitation of the heart. His legs got so bad that he could not walk at all. Several of the crew were attacked at the same time. He was on a Norwegian ship and the food consisted mainly of sotck fish. He had never had any swelling of the legs. Malaria five months ago in America.

Chest: A.C.D. normal. No murmurs. 2nd sound accentuated at Pulmonary and aortic areas. Lungs resonant and normal. Abdomen - nil.

Legs: Tenderness in thigh and claves. More marked on left side. Knee jerks absent. Plan-
tor reflex - flexor. General loss of power in legs.

Progress: Gradual improvement. Temperature and pulse normal.

Case 14.

Englishman. A.B. aged 49.

History: While in Bermuda patient one day

felt very feverish and tired and next day noticed that his legs were swelling. He felt his legs weak, and the swelling slowly spread upwards. In the course of a few days, he was swollen all over. So much so that he could not wear his own boots and clothes. He got extremely short of breath and weak and he noticed that micturition got very infrequent, passing extremely small quantities in the day and occasionally tinged with blood.

Prev. Health: Of the best.

Condition on Examination: Patient was admitted with a generalised oedema of an extreme form. His face was the only part of his body that was comparatively clear. His lower limbs extremely oedematous and tense. He is very short of breath, slightly cyanosed and hardly able to walk.

Chest Chest wall very oedematous. Pits on pressure over the sternum.

Heart: A.C.D. about Normal

Left Border: Inside nipple line

Rt. Border: Rt. Sternal margin

Apex Beat~~s~~: 5th Intrsp. inside nipple line.

Heart Sounds: closed

slight reduplic. of 1st sound at apex

Pulse: 104 per min. Regular and full - Fairly good tension. Arterial walls not thickened. Liver enlarged. Lower border about level of umbilicus.

Lungs: Dullness at both boss. Bronchitic sounds all over chest. Wheezing: prolonged expiration.

Urinary System Urine Sp.Gr. 1010 acid:

high colour. Amount in 24 hrs. 3xxii Contains a slight trace of blood and albumen.

Nervous System: K. J. present - slightly exaggerated.

Progress: 3 days after admission K.J. gone. Swelling in legs is subsiding but chest and abdomen are still very oedematous. Patient very short of breath. Heart distinctly enlarged to Rt. About 1" beyond the Rt. Sternal margin. 10 days after admission K.J. still absent - very breathless. Heart much dilated - Pulse 108 per min. Faint trace of albumen in Urine, amount 30 ozs. in 24 hours.

14 days after admission: patient much worse. Breathless and cyanosed. K.J. absent, marked Hyperaesthesia of Calf muscles. Oedema still present. Patient died suddenly through the night.

Many more notes of cases could be given to illustrate the chief clinical features, but as

the symptoms are very much the same in all the above number will suffice.

To recapitulate the symptoms and clinical features in detail, one notices that on inspection of a typical case of ship Beri-Beri the first thing that strikes one, is the appearance of exhaustion the patient presents and on enquiring what he complains of, the answer in the majority of cases is "weakness of the legs, pain in the legs and shortness of breath". This is in marked contrast to the symptoms, which according to Wright, patient with true or tropical Beri-Beri, complains of a feeling of oppression in the epigastrium, loss of appetite, fever, and general malaise and sometimes vomiting.

On further examination of the external appearances there is almost invariably at least in the first week of the disease oedema of the ankles

and legs. Pressing with the finger against the surface of the tibia produces a dimple which rapidly fills up again. Patient very often resents this procedure very much because of the pain it produces and on feeling the muscles of the calves he resents still more, there being marked Hyperesthesia in those areas.

Circulatory System:

Inspection: Cardiac impulse, sharp and rapid.

Well marked pulsation in vessels of the neck.

Palpation: Apex beats sharp, not diffuse.

May be in the normal situation but very often is displaced outwards.

Auscultation: Heart sounds as a rule closed.

Reduplication of 1st sound at apex or of 2nd in Pulmonary area. Sounds of a clicking character and quite different to what one would expect in the case of enlargement of the Cardiac area due to Hypertrophy.

Percussion: In many of the cases examined the heart dullness was within the normal limits but in the acute pernicious form of Beri-Beri with marked oedema and signs of heart failure, we find the heart as a whole dilated. The dilatation is more in the Rt. side of the heart and the Rt. ventricle is often 1-2 inches to Rt. of Rt. sternal margin. In company with this will be the usual signs of backward pressure.

Breathlessness, [∞] cyanosis

Oedema of both lung bases and dyspnoea.

Pulse: The rate of the pulse varies very much, but in cases where there is any enlargement of the cardiac dullness, the pulse is usually accelerated 80-120 beats or more per minute.

In most cases, however, it is regular in force and rhythm, but the rise and fall of the pulse wave, is very sudden, approximating very nearly the sort of pulse wave we find in aortic incom-

petence, the difference being that the rise and fall are not quite so abrupt in the former as in the latter and the apex is more sustained.

There may, or may not, be any decrotism. The arterial walls are thin and elastic unless in middle-aged individuals, who, from other causes such as alcohol, Syphilis or over-work, have some atheromatous change. The tension of the pulse therefore is low, the wave being easily obliterated. On finishing the examination of the circulatory system, one may find very little that is abnormal, or at all events not sufficient to justify one in attributing the oedema to some cardiac trouble. The tendency naturally is next to examine the urine for some abnormal constituents.

Urinary System: The amount of urine passed in 24 hours varies from 20 ozs. - 50 ozs., but in cases which are very severe and in which the

oedema comes on abruptly, the amount of urine passed is much diminished, of high colour and sp. gr., and may contain slight trace of albumen and blood. This is the exception however for in the majority of cases the sp. gr. of the urine varies from 1010-1020. The urine is clear and contains no blood or albumen. Microscopical examination of the sediment does not reveal any casts and at most only a few epithelial cells can be found.

Digestive System: According to Wright the first symptom the patient complains of, especially in the acute pernicious type, is a feeling of oppression in the stomach, tenderness in the epigastrium and nausea. I have not found this in the history of the above cases; the initial symptom complained of being loss of power and pain in the legs. There is nothing very striking in the digestive system apart from loss

of appetite, dry coated tongue and occasionally attacks of diarrhoea.

Other abdominal organs are as a rule normal, the spleen is normal in size unless the patient has a history of previous attacks of malaria. The liver is very often enlarged and congested, due probably to the backward pressure from a weakened heart.

Respiratory System: Apart from the congestion of both lung bases, there is nothing of importance to note in the respiratory system.

Nervous System:

Motor System: There is much impairment of muscular power in the lower limbs but there is seldom any actual paralysis. On bending knee on thigh and thigh on to abdomen and then asking the patient to push against your hand placed against the heel, one notices an appre-

ciable loss of power of the limb to overcome the resistance offered. This loss of power is symmetrical and as a rule is more marked in lower than upper limbs. In one case, I have seen well marked wrist drop and foot drop in both upper and lower limbs. There was no suggestion of lead palsy.

Sensations: There is often a good deal of anaesthesia in the lower limbs especially over the calf-muscles. Sensation to heat and cold is not impaired but sensation to pain is increased and this may easily be elicited when pressing the muscles of the legs, patient pulls the limb away from you or even cries out with pain.

Reflexes: The most remarkable and one of the most constant features of Beri-Beri is the absence of the knee jerks. In the beginning of the illness, the knee jerk may be exaggerated, but during the course of the illness it is in

the greater number of cases completely lost.

In a few, however, this reflex is maintained

throughout and may even continue to be exaggerated.

In the vast majority of cases, however, this tendon reflex is lost early, and when the patient comes for advice this is one of the first things that strikes one on examination. This reflex may be lost for weeks or months afterwards, and only very slowly returns as patient becomes convalescent.

Travers observations on the

Knee Jerks of 47 cases of Beri-Beri.

Period since discharge from Hospital.	No. of Cases.	Knee Jerks
13 months	1	absent
12 "	1	present
11 "	6	5 + 1-
10 "	17	16 + 1-
9 "	4	4 +
8 "	17	12 + 5-
7 "	1	1 +

In cases where the weakness and loss of power in lower limbs is marked, Rhomberg Symptom is also present, but not to such an extent as one finds it in Locomotor ataxy. The pupil reflex are normal, reacting equally to light and accommodation sensation of vision and sound are not affected as a rule. All the other cerebral functions - speech and memory, etc., are unimpaired.

Electrical reaction: During convalescence, several of the cases were tested for any signs of muscle degeneration. To the Faradic current the muscles on outer side of each thigh react, but only when the current is at its full strength. At no other points of thigh or legs was there any obvious reaction but the patient feels the current.

To the Galvanic current the muscle of thigh and legs do respond, when the current is fairly strong; those on the outer side of thigh most.

Those of legs least. Response to Cathode more readily than to anode.

The above reactions were found in typical case, which showed some wasting of the muscles after the oedema had subsided.

Haemopacitic System. The following table shows the condition of the blood in 26 cases of Beri-Beri examined by Hamilton Wright. There are no elements in the blood to indicate that the disease is one of simple or pernicious anaemia. In many of the cases of ship Beri-Beri examined in the Seamen's Hospital, the condition of the blood was practically normal; with the exception of a few cases where the disease was of few months standing the blood then showing some reduction in the percentage of Haemoglobin, according to the severity of the Anaemia.

WRIGHT'S TABLE

Blood count in 26 cases of Beri-Beri.

Sex	Stage of Disease	Type of Disease	Red Cells	White cells	Per cent Haemo-globin	Remarks
m	1 week	acute pernicious	5,430,000	7,600	92	Fatal
"	2 weeks	"	5,100,000	7,300	87	"
"	4 months	Acute	5,360,000	6,890	89	recovere
"	2 "	"	6,120,000	7,500	93	"
"	2 weeks	acute pernicious	6,103,000	7,300	98	Fatal
"	12 months	Beri-Beri Residual paralysis	4,960,000	6,800	81	Paralysi
"	3 weeks	Ac.pernici-	4,676,000	7,100	76	Fatal
"	1 week	Acute (ous	5,020,000	7,450	83	Recovere
"	2 weeks	"	5,418,000	7,400	95	"
"	2 "	Ac. pernic.	4,962,000	7,100	86	Fatal
"	7 "	Acute	5,130,000	7,600	88	Recovere
"	3 "	Beri-Beri Res. Paral.	5,280,000	7,200	91	Paralysi
"	8 months	"	5,176,000	6,980	93	"
"	6 years	"	4,896,000	6,720	87	"
"	12 months	"	4,970,000	7,600	92	"
"	10 "	"	5,670,000	7,060	92	Fatal
"	4 weeks	Ac.Pernic.	4,820,000	6,820	83	Paralysi
"	3 weeks	Acute	4,860,000	7,200	89	Fatal
"	1 week	Ac.Pernic.	5,570,000	7,100	93	Paralysi
"	1½ months	Res. Paral.	4,770,000	6,850	81	"
"	9 months	"	4,900,000	7,200	86	Fatal
"	2 weeks	Ac.pernic.	5,600,000	6,900	92	Recovere
"	3 weeks	Acute	5,000,000	7,000	89	"
"	2 weeks	"	4,600,000	7,080	86	Paralysi
"	2 weeks	"	4,780,000	6,900	89	Recovere
"	4 weeks	"	5,280,000	6,900	89	"

The chief differences in the symptoms of true Beri-Beri, and that of ship Beri-Beri are, first, the constancy in the occurrence of oedema in case of ship Beri-Beri, whereas in true Beri-Beri, oedema may or may not be present. In the latter also, Neuritis is almost invariably present, while in the former, it is often absent or very slight. In ship Beri-Beri it is rare to find paralysis during the course of the disease or as an after effect, while in tropical Beri-Beri, paralysis and even complete paraplegia is common.

See pp 30 & 31

Etiology. Various theories have been put forward as to the causation of Beri-Beri, and the facts presented by different observers are undoubtedly interesting, and serve useful ground work for further research in the hope of elucidating once and for all the mystery which enfolds the real causative agent in this disease.

Of the theories suggested only three can be

classed as worthy of earnest consideration and though the observer in each case gives many proofs in support of his own theory, the fact remains that the mystery is still unsolved.

Manson, Hamilton Wright & Braddon are the three pioneers in the field of research on this disease, and each one holds a different view to the other, in regard to the etiology . Other observers are Travers, Durham, Ross A. Davidson, Bullmore, Gelpke, Grimm, Tabaki, *Mirova* *Pekulharing* & Winkler, and many other careful workers.

Manson maintains that the disease is produced by a toxine which is either inhaled or absorbed and that this toxin, acting on the nervous system produces a well marked peripheral Neuritis. This toxin is of the same nature as that of the toxine derived from alcohol, and indeed the symptoms in both are very similar. He maintains

that the germ itself, whatever it is, is not capable of producing the disease, but given a suitable medium or locality to grow and multiply, it produces the toxine which, being absorbed or inhaled accounts for the symptoms mentioned.

In support of this Hypothesis he mentions the occurrence of Beri-Beri on ships, among Lascars & Chinese who, coming from tropical to temperate climates, feel the cold, and close windows and all ventilation, thus producing in their cabins, a stuffy vitiated atmosphere approximating in temperature as near as possible that of the Tropics.

The germ being viewed as a tropical one, has accordingly all the conditions most favourable for its growth and hence the toxin and its effect.

He also mentions an instance, reported by Hirota, which supports the view that the disease is directly caused by a toxine and not by the germ itself.

"Of 52 infants who suffered from Beri-Beri while being wet nursed by Beri-Beri mothers, 42 recovered, 5 died and 3 were not accounted for. In the cases which recovered the improvement set in at once on the case being weaned. Had a germ operating inside the body been the cause of the Symptoms the germ and the disease it produced would not have died out so rapidly, but we can readily understand the cessation of symptoms on the supposition that they were caused by the toxine which on the discontinuance of suckling was no longer being imbibed".

One would expect in such a case that the germ lived in the blood and that if a specimen of the blood were taken and cultured it would show the organism. Braddon has made very exhaustive experiment with this object in view, using strict antiseptic precautions. He tried all the dif-

ferent kinds of culture, media, acrobic and an-acrobic, but beyond Staph: albu and Flavas, nothing was grown. Blood drawn directly from a vein and cultured was absolutely sterile.

Hamilton Wright, working at the Kwala Leimpor Gaol in the Federated Malay States has made very valuable observations to disprove this theory.

He divided the number of prisoners in the Gaol into 4 parties. Party No. 1 was composed of 101 prisoners, all of whom had or had had Beri-Beri. Party No. 2 was composed of 113 prisoners who did not then have and never had Beri-Beri.

Party No. 3 was composed of 33 prisoners who at the time did not have and never had had Beri-Beri. This party was continuously added to by cases taken from party 2, who on incarceration were found to be free of signs and history of having had Beri-Beri.

Party No. 4 was composed of about 38 Cooks and tailors who had had more or less severe Beri-Beri in the gaol, but who had almost wholly recovered.

Separate rooms and sleeping berths were provided for each party and at work each party was kept isolated from the other. The diet supplied was of a uniform nature throughout, fish being excluded, and rice being fresh and free of moulds or organisms of any kind. 90 original cases of Beri-Beri developed in party No. 2. Over 100 recurrences occurred in party No. 1 and no outbreaks whatever occurred in Parties 3 & 4. Cultures from the floors and walls from Party No. 2 were attempted on ~~agar~~ rice-broth and bouillon with in many instances wholly negative results. Wright concludes by saying that the entire escape of party No. 3, let alone party No. 4 would be most unlikely in the presence of

a volatile poison that caused 90 original cases in one part and over 100 relapses in another.

Theory No. 2.

Hamilton Wright's theory.

Hamilton Wright who, as director of the Institute for Medical research, Federated Malay States, has made extensive inquiry into the Etiology and pathology of Beri-Beri, conclude that the "disease is due to a specific organism that remains dormant in certain localities, but having gained entrance to the body by the mouth, it multiplies locally, chiefly in the stomach and duodenum, gives rise to a local lesion and produces a toxin that, gaining the general circulation acts on the peripheral terminations of both afferent and efferent, ordinary and vital neurones to cause a bilateral symmetrical atrophy and that finally the organism escapes in the faeces to again lie dormant in places".

This he corroborates by showing that post-mortem appearance in case that died from acute Beri-Beri showed marked ulceration of the mucous-membrane of the Pylorus and duodenum with scattered punctiform haemorrhage on the edge of the valvulae conniventes and swollen and congested duodenal glands. This state of matters, he maintains, would account for the feeling of oppression in the gastric region with temporary loss of appetite, which, he says, are the earliest symptoms complained of in Beri-Beri. This symptom, however, was not apparent in the history of the case of ship Beri-Beri which I gave above. Neither was this pathological appearance shown in the post-mortem on some of the cases.

Theory No. 3. Braddon, another very careful observer, is very dogmatic in his view and feels positive that the disease is caused by eating

stale, or what he terms "uncured" rice. Into the Malay State provinces thousands of Chinese and Tamils or Indians are imported annually for Coolie labour. The staple food of these coolies is rice, which is of two varieties. One variety of rice is grown in Indo-China, Burma and Siam, and the other in India, chiefly Calcutta. The former Braddon regards as "uncured" the latter "cured". By "uncured" is meant, rice which after cleaning is exposed for a more or less prolonged period under the influence of air and moisture, allowing for the growths of supraphytic organisms. This is the mode of preparation of the Burma or Rangoon rice.

The cured or Indian Rice is boiled while in the husk and therefore protected from the possible spread of any specific organisms originally present on or in any of the seeds.

Braddon, in his observations, has shown de-

finitely that Tamils are fed exclusively on cured rice and the Chinese exclusively on the uncured, and ~~he~~ express the statement that "among the Chinese - the users of uncured rice - Beri-Beri is everywhere prevalent, whereas the Tamils, the users of cured rice are everywhere and always completely exempt. This is a very strong argument in favour of the Rice theory, proving almost conclusively that Beri-Beri must be due to the infection of a *fungus* or some sort of organism which grows on rice that is not cured in the manner described. The nature of the germ is not yet determined. Daniells, referring to Braddon's observations, remarks that his statements are too absolute and cites a typical case of Beri-Beri in a Tamil who had, only on very rare and exceptional occasions, a meal of uncured rice.

There are many other theories put forward with

regard to the causation of Beri-Beri, chief among these being:-

- I. Ross' theory that the cause is arsenical poisoning.
- II. Grimms' theory that it is due to the ingestion of infected fish.
- III. Gloquer's theory that it is due to a particular plasmodium on the blood.
- IV. Takaki's theory that it is due to a diminution of the nitrogenous elements in the food.

All these theories, however, have been effectively eliminated by the extensive experiments carried out by Braddon, Hamilton Wright and others.

Diagnosis

In the diagnosis of Beri-Beri (ship) one has to eliminate several other diseases, which

closely resemble Beri-Beri in their symptoms

But before doing so it might be interesting to refer to experiments quite recently performed by Azel Holst, Professor of Hygiene University of Christiania, in which he proves that a disease of the same clinical features and pathology as Ship Beri Beri can be produced experimentally on fowls, pigs and other animals fed on food prepared in a particular way.

From the fact that Ship Beri Beri all but exclusively appears on board Sailing Ships on long voyages, it is evident that the disease is very closely connected with the food. On Board these ships the food consists of raw soft Bread of inferior quality, dried biscuits, dry Baked and preserved potatoes and vegetables and tinned meat that is strongly heated before being preserved. Nocht of Norway, who was one

of the earliest advocates of food being the cause of Beri Beri, relates that in 12 out of 34 ships examined by him, in which cases of Beri-Beri occurred with sore gums and haemorrhages of the skin - and muscles, the food was of the above quality and moreover that these cases on being transferred to land, and fed on fresh animal food and green vegetables improved with marked rapidity. This is strongly in support of the argument that ship Beri-Beri is closely related to Scurvy. It may be remarked, however, that spongy gums and subcutaneous haemorrhages are comparatively rare in Ship Beri-Beri, but from the mode of onset of the symptoms, the frequency of oedema and the infrequency of neuritis, and the close relation of food to Ship Beri-Beri, it is reasonable to suppose that Tropical and Ship

Beri-Beri are two different Maladies.

Holst's experiments with strongly heated meat.

Two animals (chickens) were given ox beef boiled half an hour at 120°C. and for 1 hour at 120°C. respectively. Two control chickens got the same beef boiled for half an hour at 100°C. All boiling was done in an ordinary autoclave. The beef was always minced and found to be thoroughly boiled. The result was that all animals little by little ceased eating and lost considerably in weight. On the 79th day one of the 100° Animals was killed and one of the 120° animals was killed as a control. On the 89th day the surviving 120° animal died and on the 96th day the surviving 100° animal was killed as a control.

All animals were very emaciated. At first sight it seemed that the result of the

experiment was quite negative, that is that there was no difference between the two pairs of animals, but as a matter of fact the difference was very great.

In the first place both 120° animals showed a very marked subcutaneous oedema which did not appear in the two other chickens, In the 2nd place there was found in the two first mentioned animals a very extensive polyneuritis while the examination of the two 100° animals, was in this respect negative.

Another experiment performed on chickens fed on Meat boiled at 110° and 100° respectively. The 110° animal showed extensive neuritis while the 100° animals showed none. The evidence which Holst draws from this is that beef is able to produce polyneuritis in chickens even when boiled at a lower temperature

than 120° usually reached in the manufacture of tinned meat.

Experiment on Guinea pigs.

Guinea pigs fed on unpeeled oats, rye and barley, etc., invariably died no matter whether the grains were ground or not, at the post mortem there were pronounced haemorrhage most frequently in the muscles of the hind limbs and forelimbs and also in the muscles and the back and abdomen. There were also haemorrhages into the mucous membrane of stomach and duodenum. In some cases also into the kidneys, liver and lungs. In addition there also occurred subcutaneous oedema with pronounced fragility of the bones. Epiphysis separated from the shaft with only the intact periosteum keeping the bones in situ. In one instance the upper epiphyses of both ~~bones~~ ^{Tibiae} lay moveable on the top of the shafts and were only connected with

the latter by the periosteum. These fractures of the long bones were due to atrophy of the bone substance which was chiefly pronounced at the ends of the shafts. A pronounced fragility was also repeatedly observed in the lower maxilla. This bone sometimes actually crumbled under the fingers, also showed defects in the osseous substance.

There were also without exception looseness of the molar teeth with Hyperaemic and somewhat swollen gums. Hence, he concludes, the relationship of the disease to scurvy.

In the differential diagnoses of Ship Beri-Beri one has therefore to eliminate other causes which might account for similar symptoms but there are essential features peculiar to Beri-Beri, upon which alone the physician is able to diagnose with certainty

the nature of the malady he is dealing with.

On first seeing a typical case of Ship Beri-
Beri with Oedema of lower extremities, weakness
of the limbs, shortness of breath and all the
signs of cardiac failure, one naturally begins
with the examination of the pulse and the con-
dition of the heart. The pulse rate almost
invariably is accelerated, but regular, with
a sudden rise and fall in contrast to the
irregular, flickering pulse one would expect in
Cardiac failure from Mitral or other disease
of the heart. The area of Cardiac dullness
may or may not be enlarged, and on ausculta-
tions no definite murmur is heard. Clearly then
the condition is not due to actual disease of
the heart with subsequent Cardiac failure.

In the next place one examines the urine,
expecting to find some albumen present, but
this is conspicuous by its absence, in almost all

cases. Where it does happen to be present the quantity is so small that the examiner does not feel justified in concluding that the case before him is one of Nephritis. This is supported by the fact that there are no casts present, unless in the very acute and fatal cases where one may find a few blood casts.

The quantity of the urine may be small and variable as one gets in acute Nephritis but the specific gravity is about normal, or lower than normal. One can therefore with a fair amount of certainty exclude Renal disease.

Thirdly the Nervous system may be gone into and the first thing that strikes one here is the marked loss of power in the lower limbs. Co-ordination, however, is perfect. (It may be remarked here, however, that in convalescence from severe Beri-Beri co-ordination may be much impaired and Rhomberg Symptoms well marked.

There are no ocular symptoms however.) There may be marked Hyperaesthesia of the calves with loss of both knee Jerks. One would, of course, find this in alcoholic neuritis, or in one of the metallic poisons, such as arsenic, lead and mercury. The last three may be put out of account, as you generally find that neither of these three metals have ever been taken in sufficient quantity to produce poisoning. With regard to alcohol, one commonly finds a history of alcoholism in seamen, but neuritis from this cause is much commoner in females, who drink secretly and steadily in small quantities. At the same time one rarely meets with oedema in such a marked form in alcoholic neuritis as you do in Beri-Beri. There are no mental symptoms of any kind in Beri-Beri whereas in alcohol. mental derangements, such as delirium hallucinations and delusions are commonly met

with in conjunction with the neuritis.

After acute fevers, such as Scarlet, Diphtheria or in tuberculosis and Malignant Disease one may find a certain degree of neuritis but from the history of the case, one can safely put all these out of account.

In the nervous system therefore, there is no other disease which, on examining the case carefully and contrasting the different symptoms in each case, one is liable to mistake for Beri-Beri. There is no disease, affecting the Respiratory, Digestive or Haemoporetic systems, which in any way corresponds to this disease. Accordingly, by a process of exclusion alone, one is led to look upon Beri-Beri as a disease, distinct in itself, the unknown cause of which acts primarily on the Nervous system, producing its secondary manifes-

tations in neuritis, heart failure, oedema, etc.

PATHOLOGY

The morbid anatomy of Beri-Beri is not by any means specific. The pathological abnormalities presented vary according to the severity and the duration of the disease, and even then what one finds post mortem is not so much as one would expect in a disease that presents such definite clinical features.

The percentage of death from ship Beri-Beri is very small, and the opportunities for making post mortem examinations are not many. The following three cases, however, which died in the Seamen's Hospital, are fairly typical of the disease, but do not show any extensive lesions.

G. T. aged 49. A.B. Englishman. Admitted to the Seamen's Hospital on October 1st (See Case XIV) suffering from acute Beri-Beri. Died on the 14th October. Post mortem examination the following day.

External appearances

The body was that of a middle aged thick set man. Rigor mortis had passed off. The lower limbs were oedematous, pitting on pressure against the skin bone.

Thorax:- Left pleura adherent at apex. The pleural cavity contained half a pint of clear yellow fluid

Rt. Pleura. Rt. pleural cavity also contained about $\frac{1}{2}$ pint of clear yellow fluid.

Left Lung is very voluminous and heavy. Weighs 30 oz. There is an old. tuberculous semi-calcified nodule at apex. There is a slight amount of Emphysema at margins, and bits of the lung tissue float in water. From the cut surface much bloody oedematous fluid can be squeezed

out. The base is very dark red and friable.

Rt. Lung much the same as left. Very oedematous and bulky. Weighs 35 ozs.

Pericardium: The pericardial cavity contains a little clear fluid.

Heart: Dilated. Muscle pale and extremely friable. The finger could be pushed through the walls with ease.

The Rt. and left ventricles were much dilated and filled with semi fluid dark reddish clot, but showed no hypertrophy of walls.

The valves are all competent.

Abdomen

Liver weighed 67 ozs., is pale and soft and of a yellow wottled colour and showed fairly marked nutmeg appearance.

Spleen: Enlarged and soft with small petechial haemorrhages under the capsule.

Kidneys: Stellate veins engorged.

Pyramids very dark.

Capsule strips easily.

Pancreas: Soft and congested.

Stomach: Dilated. Mucous membrane much congested.

Duodenum: Also dilated. Mucous membrane near pylorus was a little congested. Otherwise appeared normal

Brain & Spinal Cord

Apart from excess of cerebro. spinal fluid nothing abnormal could be detected with the naked eye.

Solar plex~~us~~ and sympathetic system were dissected out but no abnormality could be made out.

Microscopical Examination

The heart muscle was specially prepared and stained with Sudd~~n~~.

Microscopic section showed fairly well marked fatty degeneration of the muscular fibres with here and there some extravasation of blood between the muscular bundles. The cardiac branches of the vagus were also carefully prepared by Merchi's method and examined but no degeneration could be made out.

Liver: showed the typical nutmeg forms, and the liver cells showed well marked fatty degeneration. The inter nobular vessels were much engorged.

Spleen: Soft and flabby. Vessels much engorged.

Kidneys: The Kidney Glomeruli were much congested. The lining membrane of the tubules showed much cloudy swelling and there was well marked inter and intratubular Haemorrhage.

Stomach Sections from mucous membrane of stomach and duodenum were examined, but beyond

marked congestion and haemorrhages, there was nothing abnormal.

Several of the peripheral nerve endings were examined and especially the Sciatic nerve. A few showed slight Wallerian Degeneration, but not to any marked extent.

The clinical notes of this case are not given along with the previous ones. Before, therefore, giving the post mortem appearances, I shall give brief notes of the clinical features.

The patient was admitted on the 30th October. He could speak no English. His condition, however was as follows: There was marked oedema of both legs, permium and Serotum. There was also oedema of thigh in Muscular and dependent parts and round the ankles.

Chest: Very thin and fairly developed with marked wasting all over.

Pulsation visible all over the pericardium
apex. Beat in 5th interspace in Mid. Clav. line.

Percussion: Upper border 3rd rib.

Left " Mid. Clav. line.

Rt. Border $\frac{3}{4}$ " to the Rt. of Rt.
Sternal margin.

Auscultation: No murmurs. Sounds very sharp
and clicking.

Pulse: Rather rapid. About 100 per min. regular.
Tension moderate. Arteries full. No
arterio Sclerosis.

Lungs: There is localised dullness at Rt.

Apex in the Supra and Infra Clav. regions.

Breath sounds harsh and prolonged with
Romchi and moist Ryles.

Left lung healthy.

Spleen: Seems to be enlarged upwards into

Axilla.. The lower border comes down to costal
margin, but the upper border extends upwards to

the 4th and 5th interspaces.

Liver. In mid axillary line commences at 4th interspace and lower border is about 2" below costal margin.

Stomach: Somewhat dilated. Lower border about $\frac{1}{2}$ " below umbilicus. There is a good deal of tenderness on palpation and patient keeps his muscle very rigid.

Nervous System.

There is loss of both Knee Jerks but plantor reflex is retained. Muscular power of both upper and lower limbs is very much impaired, but coordination is perfect.

POST MORTEM APPEARANCES

L. S. aged 38, Chinaman. The body of a medium sized Chinese appeared much emaciated. The muscles of arms and legs were small and flabby. Rigor mortis had passed off.

Thorax. Pleural on both sides very adherent and with difficulty separated from the parietes.

Lungs: Much affected with tubercle. On left side the upper lobe showed a cavity. The lower lobe a few scattered Nodules here and there.

The Rt. lung had several small cavities in upper lobe.

Heart: Small, hard and contracted. Appeared to the naked eye to be healthy but smaller than normal.

Pericardium. Healthy.

Abdomen. Liver appeared healthy

Spleen enlarged and contained several tubercular nodules.

Kidneys: Appeared healthy

The stomach and intestine seemed to be dilated.

The sciatic nerve. ~~Solar~~ plexus and sympathetic system were carefully dissected out. There seemed to be some interstitial neuritis as the nerve cords were somewhat Hypertrophied.

Microscopical Appearances.

Heart: There is no fatty degeneration in this case, but there was some degree of fatty infiltration.

Liver: Fairly healthy. No degeneration or congestion.

Kidneys. Again showed some glomerular congestion and Intertubular Haemorrhage. The lining membrane of the tubercles showed slight degree of cloudy swelling.

Stomach: Small. Did not show any congestion of the mucous membrane, either at cardiac or pyloric ends.

Spleen: Healthy

The Sciatic and several nerve terminations were cut and stained. Some of the terminal fibres showed fairly marked Wallerian degeneration.

The Sympathetic system did not show any degree of degeneration.

L. A. H. 29. Admitted July 7th

(See Case IV) Died " 9th

The body was that of a young, well developed Chinaman. There was much oedema of lower extremities.

Pleurae: Each pleural cavity contained clear serous fluid, with some recent lymph on Rt.

Visceral pleura.

Lungs: Congested. Dark red and oedematous.

Heart: Slight excess of clear fluid in the pericardium.

Rt. side of Heart dilated and full of clotted blood. Left ventricular wall slightly thickened. Valves healthy.

Liver Uniformly enlarged. Small yellow areas throughout its substance. Liver pale and fatty.

Spleen Large and firm with much fibrous tissue
Capsule thickened.

Kidneys. Capsule strips easily. To all appearance normal. No fluid in peritoneal cavity. Intestine and stomach do not show any congestion and appear to be normal.

Microscopical Examination.

The liver showed well marked fatty degeneration, with a few blood extravasations.

The Heart was fairly normal in character.

Kidneys Showed slight congestion of the Glomerular vessels and again some intertubar congestion and haemorrhage.

Intra tubular lining membrane did not show any haemorrhage or cloudy swelling.

No marked degree of degeneration could be detected in any of the peripheral nerves examined.

From the clinical History of the above cases one would expect that the pathological appearances would show more definite lesions to account for the extreme prostration with loss of power of the legs and loss of knee jerks. The fatty condition of the heart in the first case would account for the distressed feeling in the chest, the shortness of breath, and possibly to the oedema but there was no endocardial lesions.

There was no degeneration of the Cardiac nerve supply. In none of the three cases

examined were there sufficient lesion of the nervous or cardio-vascular system to account for the oedema or loss of knee jerks. The condition of the stomach and duodema which Hamilton Wright lays so much stress upon, showed in the acute oedematous cases marked congestion and blood extravasation, but this was in accordance with the condition found in all the other organs of the body, especially liver and kidneys when there is failure of the heart with backward pressure and general loss of tone of the vascular system. This loss of vascular tone is in my opinion the main cause of the oedema, but the pathological condition of the efferent and afferent system of nerves, is not in all cases sufficient to account for the loss of knee jerks. Hamilton Wright who has made exhaustive study of a case of acute pernicious Beri-Beri,

lays much importance on the morbid appearances of the stomach and duodenum.

In one case he describes "the entire epithelium of the mucosa and glandular elements as entirely altered. A large part of the cells covering the mucosa has sloughed. The outer half of the cells and of the necks and bodies of the glands which are in situ are broken down into a homogeneous necrotic mass, with an enormous amount of pigment here and there. This necrosis extends to the muscularis mucosae. The Submucosa does not exhibit necrosis or disorganization but the tissue of which it is composed appear to be dissociated - - - - Lying amongst the altered epithelial cells of the mucosa of the stomach and duodenum are a few Bacilli all of the same morphological character. They are from 4-6 M. in length, and 1 - 1.5 M. in Breadth. Their ends are slightly rounded. They

lie end to end sometimes at an angle in small colonies between the changed necrosed epithelium. They stain with Methylene Blue and are there most clearly exposed by decolorising the tissues in Aniline oil alcohol.

This organism Wright found in several of the cases examined and he thinks that its presence is certainly suggestive, and might be the cause of the gastro-duodenitis which he maintains is the primary lesion in all cases of Beri-Beri.

Wright also found in several of his cases some cellular degeneration in the Post Spinal Ganglia, and some fibre atrophy and selerosis on the Post column of the cord. This lesion, however, is very variable and not by any means constant.

TREATMENT

The treatment of ship Beri-Beri varies in individual cases and according to the stage of the disease, when the patient comes for treatment. Speaking generally the treatment varies according as to whether the case is acute, pernicious, subacute or chronic. In the acute - pernicious with marked oedema, Dyspnoea and prostration, it is of primary importance to have the patient removed from the surroundings in which the disease is supposed to have originated kept rigidly in bed, and disturbed as little as possible. Usually in this form of the disease, the pulse is accelerated, heart somewhat dilated, congestion of the base of the lungs, and of most of the organs in the body. Patient is therefore propped up in bed, and in administering medicinal treatment one aims at the reduction

of the pulse rate increasing the strength of the Systole of the heart and also at the same time to correct irregularity of the beat if there be any.

In addition one has if possible to promote diuresis, through the Kidneys or skin or by means of Cathartics and so help to get rid of the congestion and lymph Stases which is so general and prominent a feature in the acute pernicious forms of this disease.

The following is a good combination and acts well in most cases :

Rx

Tinct Digit

℥i

Pot Citras

Diuretin

aa ℥ii

Aq chloroformi ad

℥vi

Solve et Misci

℞

℥i

Every 4 hrs.

In from 30 - 48 hrs, there is a marked diminution in the pulse rate, the force of the beat is strengthened, and the heart contracts more vigorously, and this is accompanied by an increased flow of urine.

This medical treatment is continued for the first 7 - 10 days, the pulse being watched carefully in the meantime. The pulse soon comes to about normal in rate and Rhythm and any return of irregularity and acceleration of the pulse with high tension, is often an indication that the Digitalis is pushed too much. The dose must accordingly be diminished gradually. At the end of a week, in a satisfactory case, the condition and appearance of patient is vastly improved and the Dyspnoea is not so apparent. At the end of that period the medicine may be changed. In the great majority of these cases, the main cause of the anasarca and

failing heart is want of nutrition of the heart muscle and loss of power of the ventricles to contract forcibly enough to expel the blood through the system. This gradual loss of power following on want of nutrition leads to dilatation of the heart impeded circulation, with subsequent congestion and oedema. Hence, one has to study very carefully by means of the pulse, the administration of Digitalis, and not endeavour by such means to stimulate the contraction of the heart muscle, which is already from want of nutrition, too feeble to contract forcibly enough. In cases, therefore, which do not at the end of 48 hrs. show any improvement in the condition of the pulse by means of cardiac stimulants one has to resort to other means. The indication then is to give tonics and general stimulants. The following mixture acts extremely well.

R

Liq arsenici Hydroch.

Liq Strichnin Hydroch. aa 4/48

Tinct. Ferri Perchlor 3ii

Inf. Quassia ad as 3vj

M. It mist
 3i r.s. po.

This mixture acts as a nervous stimulant and at the same time improves the nutrition of the Blood, and the condition of the heart muscle. In most cases this mixture proves successful. It tends however to have a constipating effect and this must be counteracted by occasionally administering a dose of Saline purgative in the morning.

Regarding the diet in this disease it is of the utmost importance at the commencement of the treatment to give low but sustaining diets.

2 oz. of fresh milk diluted with 7 oz of Soda

water and given every 2 hrs. In addition the juice of a lemon may be given, and in the morning a cup of beef tea or Bovril.

When the pulse begins to improve and patient is gaining a little more strength the diet may be increased. For breakfast, a cup-ful of beef tea or hot milk flavoured with cocoa and some buttered toast.

For lunch fresh meat, with potatoes and green vegetables.

At tea time, some more milk and cocoa may be given and for supper light milk pudding such as rice, tapioca, etc. along with fruits.

In the severe acute cases of Beri-Beri, with extreme prostration patient requires very careful nursing and regular administration of stimulants and nourishment, and if one is successful in tiding the patient over the first two weeks of the acute stage the prognosis is very good,

and with the help of medicinal and Dietetic treatment the general loss of vitality is slowly but surely repaired, the heart beats forcibly and vigorously, the oedema disappears, and patient shows more life and takes some interest in his surroundings.

During convalescence, when patient begins to get up, massage and electricity to the limbs, night and morning, is extremely beneficial, in helping to tone up the muscles and the condition of the vascular system generally. I have found also hypodermic injections of small doses of strychnin into the lower limbs, to be very useful, when the muscles are weak, flabby and wasted.

This is injected into the calf muscles of each leg alternately once a day beginning with a small dose of Gr. $\frac{1}{80}$ and increasing it

gradually up to gr. $\frac{1}{30}$ or Gr. $\frac{1}{20}$ daily.

Patient's gait during this period is very slow and unsteady and in a good many cases well marked Rhomberg symptom is present. Patient is unable to walk along a straight line and staggers in the dark or on closing his eyes. This condition of affairs may persist for months although the patient in every other respect is in perfectly good health.

Wright classifies these cases as "Beri Beri residual paralysis". Strictly speaking there is no paralysis, the muscles only being slow in response to stimulation.

The loss of patellor reflex. is most striking and may persist for months or years, and in many cases may never return.

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